Temporal Duration and Event Size Distribution at the Epidemic Threshold

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Abstract. The epidemic event, seen as a nonequilibrium dynamic process, is studied through a simple stochastic system reminiscent of the classical SIR model. The system is described in terms of global and local variables and was mainly treated by means of Monte Carlo simulation; square lattices $N \times N$, with $N = 23, 51, 100, 151$, and 211 were used. Distinct extensive runs were performed and then classified as corresponding to epidemic or non-epidemic phase. They were examined with detail through the analysis of the event duration and event size; illustrations, such as density-like plots in the space of the model's parameters, are provided. The epidemic/non-epidemic phase presents smaller/larger relative fluctuations, whereas closer to the threshold the uncertainty reaches its highest values. Far enough from the threshold, the distribution $\phi(t)$ of the events time duration t shows a step-like appearance. However at the *threshold line* it shows an exponential behavior of the form $\phi(t) \sim \exp(-\omega t)$; the same behavior is observed for the event size distribution. These results help to explain why the approach to epidemic threshold would be hard to anticipate with standard census data.

Key words: Epidemic size, global/local variables, Monte Carlo, phase, scaling, threshold

Introduction

A wide spectrum of real world systems involves thresholds. Particularly in ecological systems, such as interacting populations or spread of diseases (Waltman, 1974), this phenomenon is very pertinent because the approach to threshold would be hard to anticipate with standard census data (Kareiva and Wennergren, 1995). The existence of a minimum value of a variable or parameter that will produce a specified effect in a particular system – commonplace in the study of critical phenomena – is often characteristic of models with increasing application in ecological systems. Many such models are characterized by critical points where sudden changes take place, with all relevant quantities of the system showing power law behavior (Kauffman, 1993).

Notably, it has been shown that many sets of real data, ranging from evolutionary dynamics of planktonic foraminifera (Stenseth and Maynard Smith, 1984) to epidemics in isolated populations (Rhodes and Anderson, 1996a,b), fall into the

class of systems that exhibit power law behavior. The mechanisms leading to the emergence of self-similar or scale-invariant patterns (power law behavior) are still not understood. One can find it in magnetic systems, for instance, but they could as well be the result of random process (Solé and Bascompte, 1996), such as random walk (Schroeder, 1991).

For epidemic systems, it is important to know if the system involves power law behavior because in this case its properties can be extrapolated from a given scale to another. However if, for instance, exponential behavior is involved, a very different situation is found: in the absence of scale invariance the natural connection between small and large events is lost.

In this work we concern ourselves with the analysis of the behavior of epidemic systems at the threshold. The general purpose is to characterize the temporal duration and the event size distribution of a stochastic model involving space and time (Renshaw, 1993) and governed by a few fundamental parameters. Because the near-threshold dynamic is not expected to essentially depend on the model's details, we use a variation of the simplest Susceptible $\rightarrow$ Infected $\rightarrow$ Removed (SIR) model (Kermack and McKendrick, 1927), which presents the threshold phenomenon. The arrows indicate that the populational exchange occurs only from susceptible to infected, and from infected to removed compartments. More complex models having not trivial dynamics are treated in another study (Aiello et al., 1998). The present model is generalized in a few different ways with respect to the original SIR model, traditionally described (Murray, 1989) by the following set of differential equations: $dS/dt = -r_0SI$; $dI/dt = r_0SI - a_0I$; and $dR/dt = a_0I$, where r_0 and a_0 are constants.

The model considered here incorporates local and global variables that can be suitably balanced. It is treated by Monte Carlo (MC) simulation method and some relevant results are illustrated through especial density maps showing nuanced patterns, and epidemic phase diagrams (Santos et al., 1998).

1. Epidemic Model

The epidemic event is considered here as a stochastic nonequilibrium dynamic process (Onstad, 1992), with each complete epidemic event being associated to one configurational path (among many possible ones) between two stable populational conformations. The dynamic evolution starts with the system in a perturbed conformation, which is precisely determined (Murray, 1989) by the susceptible population S (those individuals who can contract contagious agents $\mathcal{A}$ – biological or not), a few infectives I (those who can spread $\mathcal{A}$ to susceptibles) and a set of systemic attributes (parameters of the model). The system is closed, that is, $d[S(t) + I(t) + R(t)]/dt = 0$, where $R(t)$ is the number of removed individuals – dead or immunized individuals with respect to $\mathcal{A}$. Therefore, the number of susceptibles $S(t)$ decreases monotonically with time t , and the number of infectives $I(t)$ changes circumstantially until a new stationary conformation is reached.

The population is distributed on a 2-dimensional lattice with $N = M \times M$ sites $\{(i, j)\}$; each site is occupied by just one individual. The dynamic evolution is described by global and local variables. The global influence is treated as a mean-field effect through the mass-action assumption (Keitt and Johnson, 1995). Local interactions are taken in account by considering that a susceptible individual becomes infected with a probability that depends on the number n of infective neighbors. Therefore, the susceptible infection probability p_s , is written in the form

$$p_s = \Gamma p_G + \Lambda p_L, \quad (1)$$

where p_G is the global term and p_L is the local one. The factors Γ and Λ , satisfying $\Gamma + \Lambda = 1$, balance the effect of the global and local variable. The global term is given by $p_G = \lambda S^\mu I$, where λ and μ are real constants; λ must be $\mathcal{O}(N^{-(1+\mu)})$ to satisfy the required condition $0 \leq p_G \leq 1$. Therefore, using properly normalized $\lambda = \lambda(\mu, N)$,

$$\lambda = (1 + \mu)^{1+\mu} / (\mu^\mu N^{1+\mu}), \quad (2)$$

satisfying the constraint $0 \leq p_G \leq 1$ for arbitrary μ , we may write p_G as

$$p_G = \rho \frac{(1 + \mu)^{1+\mu}}{\mu^\mu} \frac{S^\mu I}{N^{1+\mu}}. \quad (3)$$

where $0 \leq \rho \leq 1$ limits the maximum value for p_G , and the pre-factor ρ is one of the model's parameters. The parameter μ is named *safety-in-number power* (Bailey, 1975).

As already mentioned above, the local term $p_L = p_L(i, j)$, depends on the number n of its infective neighbors $(i + \delta, j + \delta)$, with $\delta = 0, 1$ or -1 (Cardy and Grassberger, 1985). So, let p_0 be the infection probability for a susceptible when just one of its neighbors is infected. Therefore, $(1 - p_0)$ is the probability of no-infection due to just one infective neighbor, and $(1 - p_0)^n$ is the probability of non-infection due to n infective neighbors. Therefore,

$$p_L = 1 - (1 - p_0)^n, \quad (4)$$

is the probability of infection due to n infective neighbors. The classes of first and second nearest neighbors (in the square lattice) are considered as indistinguishable neighbors, and so n is an integer number varying from zero to 8.

The infective removing probability p_i is constant with respect to the time t , that is,

$$p_i = \alpha; \quad 0 < \alpha \leq 1. \quad (5)$$

The quantities ρ and α , are the main parameters of the model. In fact they are defined as probabilities per unit of time. Therefore the system is governed by p_s and p_i , and its evolution is determined by MC simulation, through the following rules:

1. one lattice site is randomly chosen;
2. if the visited site corresponds to one susceptible individual, it becomes infective with probability p_s ; otherwise, if it corresponds to one infective element, it is removed with chance p_i ;
3. now, it turns back to step (1) and the process is repeated many times, until *a priori* criterion is fulfilled (reaching the condition $I(t) = 0$).

For each site, the decision about the transition $S \rightarrow I$, or $I \rightarrow R$, is accomplished by comparing a specific generated random number $\mathcal{R}$, uniformly distributed in the interval $(0,1]$, with the corresponding probability p (p_s or p_i). Only if $\mathcal{R} \leq p$ the transition is consummated. The time unit is the MC step: $t = 1$ MC step corresponds to a complete random spanning over the lattice sites. The relationship between real time t_R and MC time t is considered below, in the next section.

2. Threshold Phenomenon

The question of threshold, for the present purpose, is summarized as follows. For simplicity we consider in this section only the global variables ($\Lambda = 0$ in Equation 1), but the effect of local variables will be also considered in our calculations.

For large N , after all sites have been visited *just once* ($\Delta t = 1$ MC-step), about $a = \rho\lambda S^{1+\mu} I$ susceptibles (Equations 2 and 3) became infected and about $b = \alpha I$ infectives (Equation 5) are removed. Note that, identifying a and b as loss rate of susceptibles and removal rate of infectives, respectively, it is straightforward to associate an amount of real time Δt_R to each MC step Δt . For systems far from equilibrium it is possible to verify, through a truly dynamical MC method (Fichthorn and Weinberg, 1991, 1992), the consistence of this coarse grained linear correspondence (Aiello et al., to be published).

The increment in the number of infectives is then represented by $\Delta I = (\rho\lambda S^{1+\mu} - \alpha)I \Delta t$, and at the beginning of the epidemic process we have

$$\left[\frac{\Delta I(t)}{\Delta t} \right]_0 = (\rho\lambda S_0^{1+\mu} - \alpha)I_0, \quad (6)$$

where S_0 and I_0 are, respectively, the number of susceptibles and infectives at $t = 0$. Since $S(t) \leq S_0$ for all $t \geq 0$, in cases that $[\Delta I(t)/\Delta t]_0 < 0$, the quantity $I(t)$ decreases systematically. Therefore we have two main trends: if $\rho\lambda S_0^{1+\mu}/\alpha > 1$, then $[\Delta I(t)]_0 > 0$ and $I(t)$ initially increases characterizing an epidemic phase; otherwise, if $\rho\lambda S_0^{1+\mu}/\alpha < 1$, the infection dies out.

The use of this criterion to decide whether the infection will spread or not is appropriated for models described by continuum variables. Clearly it cannot be employed in finite stochastic systems without precautions, because fluctuations may be significantly large. We then introduce a modification in this judgement by considering the evolution of the number of infectives $I(t)$ and its average value

$$\mathcal{I} = \frac{1}{t_c} \sum_{t=0}^{t_c} I(t). \quad (7)$$

The elapsed time t_c corresponds to the complete process, that is $I(t_c) \rightarrow 0$. We then use $I_0 = I(t = 0)$ as an upper bound: only when $\mathcal{I} > I_0$ the population is considered to belong to the epidemic phase. Therefore, small temporary fluctuations do not decide prematurely what should be the phase of the system.

3. Results

Different lattice dimensions, mainly to check how the simulation results change with the population size ($N = 23, 51, 100, 151$, and 211) were used. To construct the phase diagrams, the parameter space, described by the Cartesian product $\{\rho \times \alpha\}$, was subdivided in 100×100 small regions, with ρ and α ranging into $(0,1]$, independently of the lattice dimensions. For each pair (ρ, α) 11 MC realizations were considered, and for each one a different initial condition was used (both, slightly different value for I_0 , changing randomly about $10^{-3}N$, and a new independent sequence of pseudo random numbers).

3.1. PHASE DIAGRAMS

Figure 1a shows the phase diagram obtained, for $\mu = 0$, what corresponds to $p_G = \rho I(t)/N$ (Equation 3). A number $\xi = [0, 11]$, corresponding to the number of times the system was characterized as belonging to the epidemic phase, was associated to each pair (ρ, α) . Therefore, darker spots correspond to higher chance to find the system in the epidemic phase. Those pairs (ρ, α) with $\xi = 5$ or 6 , roughly define a boundary line $\alpha = \rho$. Note that this case, that is, $\mu = 0$ and $\Lambda = 0$, corresponds exactly to the classical *SIR* model.

Figure 1b shows the phase diagram for the case corresponding to $\mu = 1$ and $\Lambda = 0$ and $N = 100$; therefore $p_G = \rho 4S(t)I(t)/N^2$ (Equation 3). For $\mu = 1$, the fraction of the (ρ, α) plane corresponding to the epidemic region is larger then the analogous case with $\mu = 0$, since the line α vs. ρ shows a larger slope.

The results shown in Figure 1 were verified also for larger populations, $N = 151$ and 211 , but the simulations were carried out only in small regions along the threshold line $\alpha = \rho$, confirming the behavior shown for $N = 100$. Additionally, these results can be verified by direct inspection of the epidemic phase condition $\rho \lambda S_0^{1+\mu}/\alpha = 1$. For instance, for $\mu = 1$ we get $\lambda = 4/N^2$ (from Equation 2; $S_0 \cong N$) and so, $\theta = \arctan(4) \cong 76^\circ$ is the slope of the line α vs. ρ . For arbitrary μ , the *threshold region* corresponds to the neighborhood of the line

$$\alpha = [(1 + \mu)^{1+\mu}/\mu^\mu] \cdot \rho. \quad (8)$$

Figure 2 shows, for $N = 100$, the effect of the local variables on the phase diagram. The labels a , b , and c correspond, respectively, to $\Lambda = 0.2, 0.5$ and 0.8 ;

Figure 1a. Threshold phenomenon; global variables only. Phase diagram with α and ρ varying from 0.01 to 1 by steps $\delta = 0.01$. Black and white regions correspond, respectively, to epidemic and non-epidemic phases. At the threshold region, $\rho \approx \alpha$, darker spots mean higher chance for the infection to spread out. (a) $\mu = 0$: the threshold phenomenon is revealed by the transition dark-clear region, which, besides fluctuations, clearly indicates the boundary line $\alpha = \rho$. This result represents the exact solution of the original SIR model. (b) Same as in (a) but now for $\mu = 1$: note that the slope of the threshold line has increased from $\theta = 45^\circ$ to $\theta \cong 76^\circ$: $\alpha = \rho$ (see Equation 3).

the same $p_0 = 0.05$ and $\mu = 0$ was used in all three cases. Accordingly, as Λ approaches to one, the dependency of the *threshold line* on ρ becomes smaller. Of course, in the limit $\Lambda \rightarrow 1$, (only local variables) the threshold line approaches to $\alpha = C$, with C depending only on p_0 , that is, $C = C(p_0)$; $C(0) = 0$. The sequence of results shown in Figure 2 can be understood by analyzing separately the superposition of the two terms of the right-hand side of Equation 1. Let us therefore consider first the term Γp_G . The factor Γ may be considered as acting directly on the parameters ρ , corresponding to the linear transformation $\rho' = \Gamma\rho$ in Equation 8. Therefore, the threshold line is then described by $\alpha = [(1+\mu)^{1+\mu}/\mu^\mu] \cdot (\Gamma\rho)$.

On the other hand, the multiplicative factor Λ acting on p_L reduces p_0 (Equation 1 and 4); recall that $\Lambda \leq 1$. For sufficiently small p_0 , we have $p'_0 \cong \Lambda p_0$. Therefore, the resulting threshold line, involving local and global variables, and for arbitrary μ , can be expressed in the form

Figure 1b.

$$\alpha = [(1 + \mu)^{1+\mu} / \mu^\mu] \cdot \Gamma \rho + C(\Lambda p_0); \quad p_0 \ll 1. \quad (9)$$

3.2. EPIDEMIC BEHAVIOR AT THE THRESHOLD

Our numerical analysis of the temporal behavior considers initially only the global variables by setting $\Gamma = 1$ and $\Lambda = 0$. The Figure 3 gives a general view of the average event duration $\langle t \rangle$, as a function of (ρ, α) , with $\mu = 0$ and for $N = 100$; darker areas correspond to longer event duration. For any fixed ρ , $\langle t \rangle$ decreases as α increases from zero. Approaching the threshold line, $\alpha \cong \rho$, it increases back, producing nuanced patterns on the lower triangular region of the (ρ, α) plane. For all $\alpha > \rho$, $\langle t \rangle$ quickly decreases back. Here also, for each pair (ρ, α) , $\eta = 11$ independent MC realizations were considered in the calculation of the average $\langle t \rangle = \sum t_i / \eta$.

The uncertainty on the epidemic duration data was estimated by the relative dispersion of the MC time data (normalized standard error of the mean). For each one of the 10 000 points (ρ, α) , the sample variance $\delta^2 = \sum_i (\langle t \rangle - t_i)^2 / (\eta - 1)$ and the uncertainty, defined as $\Delta = \delta / (\sqrt{\eta} \langle t \rangle)$, were calculated. Figure 4 shows that the smaller uncertainties are found in the epidemic phase – clear region. The non-

Figure 2. The effect of the local variables on the phase diagram; $p_0 = 0.05$ was used. In (a) the local variable influence is small, $\Lambda = 0.2$; in (b) it is even balanced with the global variables, that is, $\Lambda = 0.5$; and in (c) the local influence is predominant: $\Lambda = 0.8$. Darker spots, as in the previous figures, are related with a higher chance to find the system in the epidemic phase.

Figure 3. Average epidemic event duration $\langle t \rangle$; global variables only and $\mu = 0$. Small values for α give long time duration (trivial correspondence to the slow removing of infectives). However, long time duration is also observed in the threshold region, $\rho \approx \alpha$.

epidemic phase – upper triangular region – shows larger Δ , but in the threshold region, $\rho \cong \alpha$, the relative fluctuation reaches its highest values. This large uncertainty means that small perturbations on the initial conditions may produce large differences on the epidemic process. Far enough from the threshold, the system is much less sensitive.

Although depending on the initial conditions, as mentioned above, at the threshold the epidemic event duration t is not correlated with its corresponding initial number of infectives $I_0 = I(t = 0)$. The absence of significant correlation was verified by calculating, for $\mathcal{N} = 400$ specific and independent events, the following test

$$\mathcal{C}(t, I_0) = \frac{1}{\mathcal{N}} \sum_{i=1}^{\mathcal{N}} \text{sign}(t_i - \langle t \rangle) \cdot \text{sign}(I_{0,i} - \langle I_0 \rangle), \quad (10)$$

Figure 4. The uncertainty Δ is larger in the non-epidemic phase (gray background – upper triangular region) than in the epidemic phase. However, the largest uncertainty is found on the threshold region.

where i labels the event; $\text{sign}(x)$ means signal of x ; $I_{0,i}$ is the initial number of infectives and t_i is its respective event duration, with respect to the i th event. The averages $\langle t \rangle$ and $\langle I_0 \rangle$ were calculated over all 400 events considered. The small numerical value found, $\mathcal{C} \cong 0.16$, indicates a random dependence between t and I_0 .

The inclusion of local variables ($\Lambda \neq 0$; Equation 1 and 4) does not change qualitatively the results discussed above, with respect to the temporal behavior at the epidemic threshold. Figure 5 illustrates its effect on the time duration. The pair $\rho = 0.2$ and $\alpha = 0.3$, corresponding initially to a non-epidemic phase, is fixed, and Λ changes systematically from zero. The critical value $\Lambda = \Lambda_c$ ($\Lambda_c \cong 0.1$ for the present case, with $p_0 = 0.3$) separates two phases: for $\Lambda > \Lambda_c$, the system gets through the epidemic phase.

Now, organizing our results by counting the number $\mathcal{N}(t > \tau)$ of epidemic events of duration $t > \tau$, a random-like phenomenon at the epidemic threshold

Figure 5. The local variables effects. The parameters $\rho = 0.2$, $\alpha = 0.3$ and $p_0 = 0.3$ are fixed, and Λ changes systematically from zero (for $\Lambda = 0$ it corresponds to non-epidemic phase). Depending on p_0 , there is a threshold value $\Lambda = \Lambda_c$ ($\Lambda_c \cong 0.1$ for $p_0 = 0.3$); for values above Λ_c , the system is in the epidemic phase. In (a) it is shown the number ξ of times the system was found in the epidemic phase, as function of Λ ; (b) shows that the average event duration is significantly larger in the threshold region. The connected empty circles are the numerical results smoothed by the thick continuous curve.

became fully apparent. As it is shown in Figure 6a, at the threshold ($\alpha = \rho = 0.3$ for this case), $\mathcal{N}$ has an exponential dependence of the form $\mathcal{N}(t > \tau) = \exp(-\omega\tau + a)$, where a is a constant. Therefore the frequency ϕ of events with duration t is given by $\phi(t) = [\partial\mathcal{N}(t > \tau)/\partial\tau]/\partial\tau|_{\tau=t} \sim \exp(-\omega t)$, and the epidemic event lifetime $\int_0^t t' \phi(t') dt' \equiv \langle t \rangle = \omega^{-1}$. Indeed, the epidemic event lifetime ω^{-1} depends on the population size. It increases with N but changes sluggishly for large enough populations ($N > 150$), as suggested by Figure 6b.

Another way to interpret $\mathcal{N}(t > \tau)$ can be achieved by considering an ensemble of $\mathcal{N}_0$ independent epidemic events, with all events starting at $\tau = 0$; $\mathcal{N}_0 = \mathcal{N}(t > 0)$. Therefore, $\mathcal{N}(t > \tau) = \int_0^\tau \phi(t) dt$ is the number of systems that at time τ is still running an epidemic process. The decay constant ω is the *a priori* probability per unit of time for a particular epidemic event to stop after any time $t > 0$.

Figure 7 shows the behavior of $\mathcal{N}(t > \tau)$ far from the threshold line. Either, in the epidemic ($\rho = 0.3$, $\alpha = 0.2$) or non-epidemic ($\rho = 0.3$, $\alpha = 0.4$) phase, $\mathcal{N}(t > \tau)$ presents a step-like function behavior, what implies in a localized event duration distribution. Therefore, we may say that far enough from the threshold, the epidemic time behavior is quite deterministic.

The same analysis, managed for the epidemic size data, revealed equivalent random-like behavior. We just illustrate it, in Figure 8, through the number $\mathcal{N}(s > \sigma)$ of events with size s larger than σ . The size s measures the total number of infectives removed.

Figure 6. Random-like behavior at the threshold. Part (a) shows the number $N(t > \tau)$ of events with duration t larger than τ . An exponential $\exp(-\omega t + a)$, thin continuous line, fits quite well with the data. The part (b) shows how the epidemic event lifetime ω^{-1} depends on the population size $N = 23, 51, 100, 151$, and 211 were used. The lifetime ω^{-1} increases with N but changes slowly for large enough ($N > 150$) populations. The continuous line fitting the data was introduced as eye guide.

Figure 7. Temporal behavior far from the threshold. In curve (a), $\rho = 0.3$; $\alpha = 0.2$, and in (b), $\rho = 0.3$; $\alpha = 0.4$, belonging, respectively, to epidemic and non-epidemic phase. This step-like function behavior indicates that far from the threshold the event time duration is quite deterministic (compare with Figure 6a).

4. Final Comments and Conclusion

In this work the epidemic event is considered as a stochastic nonequilibrium process. It starts with the system in a perturbed conformation, determined by a susceptible population and a few infectives, and stops when a new equilibrium conformation is found. A generalization of the original *SIR* model, involving only single epidemics, is employed with the main purpose of fully characterizing the time duration and event size of the epidemic process near to the threshold. Global influence on the population was considered as a mean-field effect. Local interactions were also considered by means of the number of effective contacts (first neighbors) among each susceptible and its infective neighbors. The system was treated by means of Monte Carlo simulations, using square lattices $N \times N$ of different sizes to represent the population; $N = 23, 51, 100, 151$ and 211 were used. However, due to computational cost, the results were illustrated mostly using $N = 100$.

Epidemic and non-epidemic phases were determined and illustrated through density maps in the parameter space. A formal solution for the threshold line, involving all the model's parameters (namely: μ , Γ , Λ , α , ρ and p_0) was also derived (Equation 9). The consistency of our approach was tested by comparing the result for specific values for the model's parameters, namely $\mu = 0$ and $\Lambda = 0$, with that corresponding to the original *SIR* model.

Far enough from the threshold, the event duration is well defined, characterizing a deterministic nonequilibrium phenomenon. For all experiments considered, the

Figure 8. Number $N(\sigma > s)$ of events with size σ larger than s . The size σ measures the total number of infected individuals during the epidemic event. At the threshold, the same random-like behavior, as that shown in Figure 6a, is observed.

event duration was not sensitive with respect to the initial conditions, giving a high degree of predictability for the epidemic process.

Near to the threshold the epidemic process resembles the typical microscopic behavior of a physical system at equilibrium, when interactions among units are not so important and random phenomena are more relevant. Indeed, a slight change in the initial condition (random distribution of a few infectives) may, or may not induce a large change in the event duration or event size. This means that initial and final population conformations are not correlated: the system has no memory. So, at the epidemic threshold, the uncertainty about the epidemic process became significantly larger.

It is relevant to know, for epidemic systems, if a particular system involves power law behavior or not because, for practical purpose, it is important to make extrapolation from a given scale to another to make connections between small and large events. Then, in order to verify what kind of behavior the system studied here presents, an additional extensive analysis, involving 400 MC realizations, for a single pair (ρ, α) at the threshold was carried out. It determined that the frequency ϕ of events with duration t has an exponential-like form, $\phi(t) \sim \exp(-\omega t)$. This

was achieved by counting the number $\mathcal{N}(t > \tau)$ of epidemic events of duration $t > \tau$, for different values of τ . The same behavior was found for the epidemic event size. Therefore, at the epidemic threshold, the best that one can say about the event duration (or event size) is that it has a lifetime $\langle t \rangle = \omega^{-1}$.

For cases like this, presenting purely stochastic behavior, it is improbable to recognize (using only standard census data) the imminence of any epidemic disaster if the system is near to the threshold region.

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